Quantitative Trait Loci for Antibiosis Resistance to Corn Earworm in Soybean

B. G. Rector,* J. N. All, W. A. Parrott, and H. R. Boerma

ABSTRACT

In more than 25 yr since the discovery of soybean [*Glycine max* (L.) Merr.] resistance to defoliating insects, attempts to introgress this trait into elite germplasm have been relatively unsuccessful. Resistance to defoliating insects in soybean is expressed as a combination of antibiosis (toxicity) and antixenosis (nonpreference). Both of these resistance modes are inherited quantitatively in soybean. The objectives of this study were (i) to use restriction fragment length polymorphism (RFLP) maps to identify quantitative trait loci (QTLs) in soybean for antibiosis against corn earworm (CEW) (*Helicoverpa zea* Boddie), (ii) to determine the relative magnitude, gene action, and genomic locations of these QTLs, and (iii) to compare them to previously detected soybean antixenosis QTLs. Restriction fragment length polymorphism maps were constructed in three soybean F_2 populations segregating for antibiosis against CEW: 'Cobb' × PI171451, Cobb × PI227687, and Cobb × PI229358. Antibiosis was measured as larval weight gain in a detached leaf assay. The RFLP data were associated with insect bioassay data to detect QTLs for antibiosis in each cross. Variance component heritability estimates for antibiosis in the three crosses were 54, 42, and 62% in Cobb × PI171451, Cobb × PI227687, and Cobb × PI229358, respectively. An antibiosis QTL on Linkage Group (LG) M was detected in both Cobb × PI171451 and Cobb × PI229358 (R^2 values of 28 and 22%, respectively). An antixenosis QTL was also significant at this location in the same two crosses. This was the only insect-resistance QTL that was detected for both antibiosis and antixenosis. Antibiosis QTLs were also detected on LGs F and B2 in Cobb × PI227687 (R^2 = 33 and 12%, respectively), and LGs G and J in Cobb × PI229358 (R^2 = 19% for each). Antibiosis was conditioned by the PI (resistant parent) allele at the QTLs on LGs G, M, and B2, whereas the susceptible parent, Cobb, provided antibiosis alleles at the QTLs on LGs F and J.

Plant resistance to insects (PRI) has been a difficult trait for soybean breeders to efficiently introgress into elite cultivars. In more than 25 yr since high levels of PRI were discovered in the soybean plant introductions PI171451, PI227687, and PI229358 (Van Duyn et al., 1971), only three soybean cultivars have been released with these PIs in their parentage (Bowers, 1990; Hartwig et al., 1990, 1994). None of these cultivars combines high levels of PRI with competitive yields and none is popular among soybean growers today. Insect resistance in these PIs has been demonstrated against a number of defoliating insect species (Luedders and Dickerson, 1977; Lambert and Kilen, 1984a; All et al., 1989) including the corn earworm, a common pest of soybean in the southeastern USA. Resistance against CEW has been closely correlated with other major soybean pests including the soybean looper (*Pseudoplusia includens* Walker) (All et al., 1989).

There are several reasons for the difficulty experienced in breeding for PRI in soybean. Resistant germplasm is of low agronomic quality. Insect resistance is inherited quantitatively in all three resistant PIs (Sisson et al., 1976; Luedders and Dickerson, 1977; Rufener et al., 1989; Kenty et al., 1996), making full introgression difficult and increasing the potential for inferior yield through linkage drag (Zeven et al., 1983; Young and Tanksley, 1989). Resistance in these PIs is expressed through two distinct mechanisms; antibiosis (Painter, 1951; Lambert and Kilen, 1984a) and antixenosis (or nonpreference) (Clark et al., 1972; Kogan and Ortman, 1978). Antibiosis describes insect resistance in which feeding on the plant results in mortality or disruption of growth, development, or physiology in the insect. Antixenosis, or nonpreference, describes resistance in which the insect is either repelled from or not attracted to its normal host plant. Antibiosis and antixenosis can be assayed separately, although their effects may overlap (i.e., an antibiotic chemical may also repel). Soybean breeding programs which have worked with PRI have primarily selected lines based on assays for only one of the two resistance mechanisms, perhaps under the assumption that they are genetically indistinct. However, the genetic independence of antibiosis and antixenosis in PRI has been suggested for some time (Painter, 1951; Manglitz and Danielson, 1994). Through the detection of QTLs contributing to antibiosis against defoliating insects in soybean and comparison of such QTLs to those previously detected for soybean antixenosis (Rector et al., 1998, 1999), we will attempt to show that the failure of efficacious introgression of PRI into soybean cultivars may be due in part to the lack of selection for both resistance components.

Quantitative traits can be dissected into their individual Mendelian components by statistically associating their inheritance with that of markers on a genetic map (Thoday, 1961; Paterson et al., 1988). The advent of DNA marker technology in genetic mapping (Botstein et al., 1980; Williams et al., 1990; Akkaya et al., 1992; Vos et al., 1995) has allowed the construction of saturated genetic maps in many crop species (O'Brien, 1993) including soybean (Shoemaker and Specht, 1995; Keim et al., 1997; Cregan et al., 1999), and the identification of QTLs for a variety of agronomically important traits including CEW antixenosis resistance in soybean (Rector et al., 1998, 1999). Molecular markers have also been

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Abbreviations: AFLP, amplified fragment length polymorphism; CEW, corn earworm; cM, centiMorgan; LG, linkage group; LOD, logarithm of the odds; PI, plant introduction; PRI, plant resistance to insects; QTL, quantitative trait locus; RFLP, restriction fragment length polymorphism; SSR, simple sequence repeat; USDA/ISU, United States Department of Agriculture/Iowa State Univ.

employed to study the quantitative inheritance of PRI in several other crop species including maize (*Zea mays* L.) (Byrne et al., 1997), mungbean (*Vigna radiata* L. Wilczek) (Young et al., 1992), potato (*Solanum tuberosum* L.) (Yencho et al., 1996), and tomato (*Lycopersicon esculentum* Miller) (Maliepaard et al., 1995), although none of these studies sought to specifically compare antibiosis and antixenosis QTLs.

The objectives of this study were (i) to identify genetic markers associated with QTLs for antibiosis in soybean PI171451, PI227687, and PI229358; (ii) to determine their relative magnitudes, gene action, and genomic locations; and (iii) to compare them to markers previously associated with antixenosis QTLs in soybean (Rector et al., 1998, 1999).

MATERIALS AND METHODS

Restriction fragment length polymorphism maps were constructed using F_2 populations of crosses between an insect-susceptible soybean cultivar, Cobb, and three insect-resistant soybean plant introductions: PI171451, PI227687, and PI229358. The mapping populations for Cobb × PI171451 (Cross 1), Cobb × PI227687 (Cross 2), and Cobb × PI229358 (Cross 3) consisted of 110, 95, and 103 F_2 plants, respectively. The F_2 plants from the three crosses were derived from seven, eight, and four F_1 plants, respectively, and were grown at the University of Georgia Plant Sciences Farm near Athens, GA in 1993. Leaves were harvested from F_2 plants for DNA isolation. At maturity, seeds from each F_2 plant were bulked to create $F_{2:3}$ lines.

DNA isolation, Southern blotting, and hybridization procedures were performed as previously described by Lee et al (1996). Approximately 400 probes from various sources, including cDNA and genomic clones of soybean and other cultivated legumes, were used to screen for RFLP between Cobb, PI171451, PI227687, and PI229358. Five restriction enzymes (*Dra*I, *Eco*RI, *Eco*RV, *Hind*III, and *Taq*I) were used to identify RFLP. Polymorphic probes were used for genetic mapping. Genetic linkage was determined using the computer mapping software MAPMAKER/EXP (Lander et al., 1987) using the Haldane mapping function with a minimum logarithm of the odds (LOD) score of 3.0 to establish linkage at a maximum distance of 50 centiMorgans (cM). All named LG on the maps of all three crosses were anchored to the USDA-ARS/ISU map based on the matching of RFLP allele enzyme and band-size information from both parents of each cross (data not shown). Linkage group names correspond with those found on the recently integrated soybean simple sequence repeat (SSR)/RFLP map (Cregan et al., 1999).

A larval weight gain assay, adapted from a previously described technique (Lambert and Kilen, 1984a), was employed to measure antibiosis. In each of our three populations, six plants from each $F_{2:3}$ line and each of the two parents were grown to the V4 growth stage (Fehr and Caviness, 1977) in a 1-L styrofoam cup under a 15:9 h (light/dark) photoperiod to prevent flowering. Leaflets from the most recent fully expanded trifoliolate of each greenhouse-grown V4 plant were detached for insect infestation. An experimental unit consisted of one leaf from each of the six plants that represented an $F_{2:3}$ or parental line. Three neonate CEW larvae were placed on the detached leaflets which were kept turgid with moistened filter paper and sealed inside petri plates with parafilm. Larvae were allowed to feed until the leaves in any one of the petri plates were completely consumed. The experiment was replicated three times in a randomized complete block experimental design for Crosses 1 and 2 (a total of 18 leaves tested in each $F_{2:3}$ line) and four times for Cross 3 (24 leaves tested in each $F_{2:3}$ line).

In initial experiments (data not shown) three different parameters, larval mortality, larval weight gain, and larval head capsule width, were tested to measure antibiosis against CEW. Weight gain and mortality differed significantly ($P < 0.05$) between the parents of all three crosses, while head capsule width differed only between Cobb and PI229358. Based on differences between the parents of each cross and preliminary data in $F_{2:3}$ lines, larval weight gain was chosen to measure of antibiosis for this experiment.

After feeding, larvae were frozen and weighed. The total weight of all surviving larvae was recorded for each dish. The mean of the total larval weights from each line was calculated for each replicate and least square means for each line were calculated to account for missing data (PROC GLM; SAS Institute, 1988). Variance component heritability estimates (h^2) were calculated for each population from $F_{2:3}$ family means based on the same selection unit used in the larval weight gain assay (Fehr, 1987). Least square mean data were associated with RFLP marker data to detect marker–QTL linkage. Cosegregation of RFLP markers with antibiosis QTLs was detected by interval mapping (Lander and Botstein, 1989) with the computer program MAPMAKER/QTL (2.0 LOD threshold; Lincoln et al., 1992). The LOD score peak was used to estimate the most likely QTL position on the RFLP linkage map. The percentage of variance explained by each QTL (R^2) and the additive (a) and dominance (d) effects were estimated at each maximum likelihood QTL peak using the TRY function of the MAP command in MAPMAKER/QTL. The average degree of dominance for each QTL was calculated as the ratio d/a and this value was used to estimate the gene action (e.g., additive, dominant, recessive) of a QTL. Marker–QTL relationships were confirmed using a general linear model (PROC GLM; $P < 0.01$; SAS Institute, 1988) in which the genotypic class (i.e., AA, Aa, aa) of the RFLP marker was the predictor variable and the mean larval weight gain data was the response variable. Markers which did not display normal segregation (1:2:1) were not accepted ($X^2 < 0.05$) as significant marker–QTL associations. Alignment of linkage groups in comparative mapping was contingent upon linkage groups sharing markers which were mapped using the same restriction enzyme and restriction fragment. Multiple regression analysis was performed on all significant markers within each cross to estimate total phenotypic variance explained.

RESULTS AND DISCUSSION

Restriction fragment length polymorphism maps were constructed in Crosses 1, 2, and 3. The RFLP map in Cross 1 had 85 markers on 21 LGs with 15 markers which did not link to any others. This map covered 1113 cM. In Cross 2, the RFLP map had 120 markers on 26 LGs with 13 unlinked markers. This map had 1470 cM. The Cross 3 map had 129 RFLP markers on 30 LGs with 10 unlinked markers and 1566 cM.

In Cross 1, there were small, isolated areas of abnormal RFLP marker segregation that were localized to specific regions of several linkage groups. Certain $F_{1:2}$ families did not segregate at all for these markers and one $F_{1:2}$ family segregated 1:1, while three of the seven $F_{1:2}$ families (approximately half of the total number of F_2 individuals) showed a normal 1:2:1 segregation. For these markers, only the data from normally segregating

$F_{1:2}$ families were used for mapping purposes and none of these markers were accepted as significant in QTL detection. The antibiosis data suggest that there is no correlation between this abnormal segregation and the incidence of insect resistance QTLs. No such abnormal regions were detected in Crosses 2 or 3.

Transgressive segregation for antibiosis was observed in each of the three crosses (Fig. 1). In Cross 1, the mean larval weight gain for larvae fed on Cobb and PI171451 was 18.8 and 6.2 mg, respectively. The range among the F_2-derived lines was from 2.4 to 31.4 mg. In Cross 2, the means for Cobb and PI227687 were 49.6 and 33.3 mg, respectively. The range in this cross was from 15.2 to 84.0 mg. In Cross 3, the means for Cobb and PI229358 were 16.3 and 5.7 mg, respectively, and the range among the F_2-derived lines was from 2.2 to 22.7 mg. The larger weights in Cross 2, relative to Crosses 1 and 3, were due to an unexpected rise in laboratory temperature during the Cross 2 experiment. However, the Cross 2 parent means were significantly different and the population distribution was qualitatively similar to that of Crosses 1 and 3 (Fig. 1). Therefore, the data were considered suitable for QTL analysis.

A QTL for antibiosis was detected in the interval between markers A584V and A226H-1 on LG M in both Crosses 1 and 3 (Table 1). These markers were polymorphic and mapped to the same genomic location in Cross 2, but were not associated with insect resistance in that cross (Fig. 2). This QTL accounted for 28% of the phenotypic variance (R^2) for antibiosis in Cross 1 and 22% of the phenotypic variance for antibiosis in Cross 3. Heritability estimates (h^2) for this trait in Crosses 1 and 3 were 54 and 62%, respectively. In both crosses, resistance was provided by the PI allele and was partially recessive (Table 1). Marker A584V was also associated with a major antixenosis QTL in Crosses 1 and 3, but not Cross 2 (Rector et al., 1998, 1999). It is possible that it is the same QTL that is being detected with both the antibiosis and antixenosis bioassays.

Two more QTLs were detected in Cross 3 (Table 1). They were in the interval between markers L183H and L002H on LG G and between markers A064V and K401H on LG J. The QTL on LG G explained 19% of the phenotypic variation for antibiosis in this cross. Resistance is conditioned by the PI229358 allele and is partially dominant. The QTL on LG J also explains 19% of the phenotypic variation for the trait. Resistance at this QTL is conditioned by the Cobb allele and is dominant.

Two QTLs for antibiosis were detected in Cross 2 (Table 1). These were on LG F, in the interval between markers A083I and Cr207V, and on LG B2, between markers A343V-2 and K411T-1. They accounted for 33 and 12% of the phenotypic variance for antibiosis in Cross 2, respectively. The heritability estimate for antibiosis in this cross was 42%. Resistance from the QTL on LG F is conditioned by the Cobb allele and is dominant, whereas resistance from the QTL on LG B2 is provided by the PI227687 allele and is partially dominant.

Attempts were made to associate the antibiosis QTLs

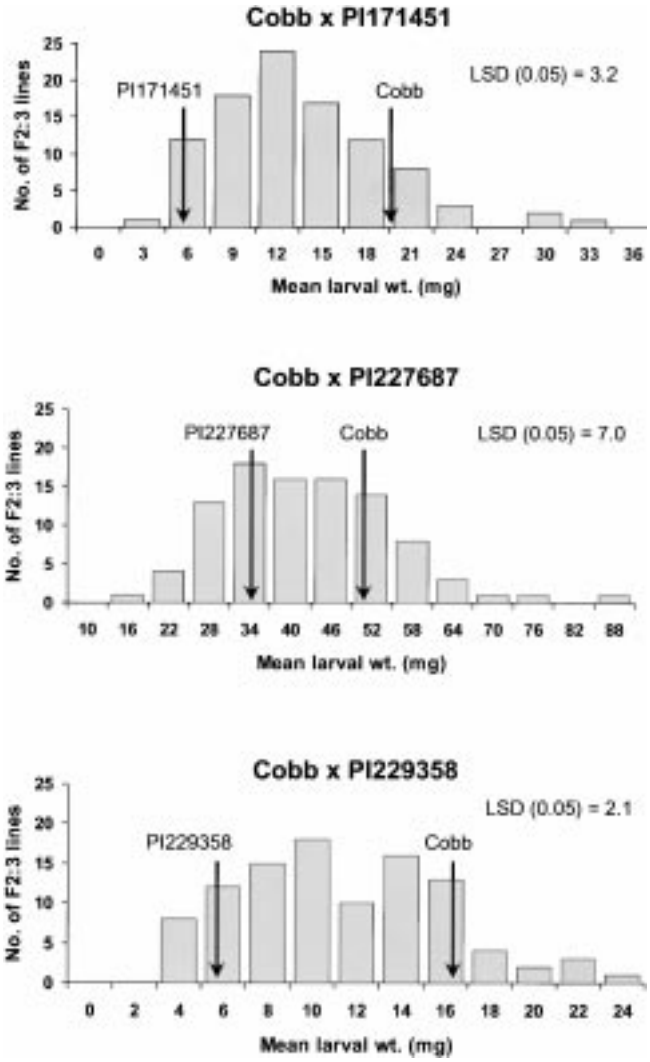

Fig. 1. Distribution of F_2-derived lines of the crosses Cobb × PI171451, Cobb × PI227687, and Cobb × PI229358 based on mean larval weight gain of corn earworm fed on these lines.

detected in this study with insect resistance genes that have been reported in mungbean (Young et al., 1992) and cowpea [*V. unguiculata* (L.) Walpers] (Myers et al., 1996). Insect resistance genes in these two legume species could not be associated with any of the linkage groups containing soybean antibiosis QTLs, based on comparative mapping between soybean and mungbean (Boutin et al., 1995) and between soybean, mungbean, and cowpea (Menancio-Hautea et al., 1993).

The only QTL for antibiosis that was detected in more than one genotype was the QTL linked to marker A584V on LG M (Table 1). This was also the only marker that was associated with both antibiosis and antixenosis (Rector et al., 1999). All other soybean insect resistance QTLs that have been detected condition either antibiosis or antixenosis but not both (Rector et al., 1998, 1999). It is interesting to note that antibiosis at this QTL is partially recessive (Table 1), while antixenosis at this QTL is inherited additively (Rector et al., 1999). This may reflect a qualitative difference in the two bioassays or the presence of two separate QTLs,

Table 1. Restriction fragment length polymorphism (RFLP) markers associated with antibiosis against corn earworm in three soybean populations, based on MAPMAKER/QTL analysis.

		Cobb × PI171451 (Cross 1)					Cobb × PI227687 (Cross 2)					Cobb × PI229358 (Cross 3)				
Map interval (cM)	Linkage group	QTL pos.†	LOD	R^2	*a*‡	*d*/*a*§	QTL pos.†	LOD	R^2	*a*	*d*/*a*	QTL pos.†	LOD	R^2	*a*	*d*/*a*
				%					%					%		
A343V-2..K411T-1 (30.6)	B2	–	NS¶	–	–	–	1.5	2.2††	12	−6.4	0.25	–	NS	–	–	–
A083I..Cr207V (46.8)	F	–	NP#	–	–	–	28.0	3.8††	33	7.6‡‡	1.27	–	NP	–	–	–
L183H..L002H (13.2)	G	–	NP	–	–	–	–	NP	–	–	–	6.0	3.8††	19	−2.7	0.37
A064V..K401H (40.8)	J	–	NS	–	–	–	–	NS	–	–	–	34.0	2.8††	19	2.6‡‡	−0.01
A584V..A226H-1 (23.8)	M	8.0	5.0††	28	−3.5	−0.87	–	NS	–	–	–	1.5	4.8††	22	−2.9	−0.45

† Distance of QTL peak from first marker listed.
‡ Average change in larval weight gain for each PI allele.
§ Average degree of dominance for each allele; *d* = dominance effects, *a* = additive effects.
¶ NS = interval was polymorphic but not significantly associated with antibiosis.
# NP = no polymorphism was detected at this interval in this cross.
†† Significance confirmed with analysis of variance ($P < 0.01$).
‡‡ Resistance allele from Cobb.

perhaps in a gene family. Computer simulations using small populations (100 F_2 progeny) to search for QTLs with small effects (<10% R^2 per QTL) suggest that errors in estimating variance explained and degree of dominance can be great (Beavis, 1998). Thus, the smaller QTL detected in Cross 2 could be a false positive, and comparisons of QTL gene action between populations should be made with caution.

Two antibiosis QTLs, on LG 'F' in Cross 2 and on LG 'J' in Cross 3, derived resistance from the Cobb (susceptible parent) allele. In Cross 2, resistance comes from the Cobb allele at a QTL between markers A083I and Cr207V (Table 1; Fig. 2). Both PI171451 and PI229358 share the same RFLP alleles as Cobb for these two markers (data not shown), so it is possible that these genotypes also share the resistance allele at the QTL.

The resistance allele from Cobb on LG J was detected 6.8 cM from marker K401H in Cross 3. This region of LG J is represented in both Crosses 1 and 2 (Fig. 2). In Cross 1, the region was not considered because it is flanked by two markers, K401H and A458V, which both showed abnormal segregation (see above). In Cross 2, a subthreshold LOD peak (1.98) is evident at marker A963I-2, which is 7.7 cM from K401H. The *F* test at this marker was also just below threshold ($P = 0.014$). These data, combined with the mean larval weight data for the three genotypic classes at this marker (data not shown), suggest that the Cobb resistance allele may be evident in this cross as well, but with a smaller effect than in Cross 3. The difference in detection at this locus may also be due to inadequate sampling of the gamete pools due the small population sizes (Beavis, 1998).

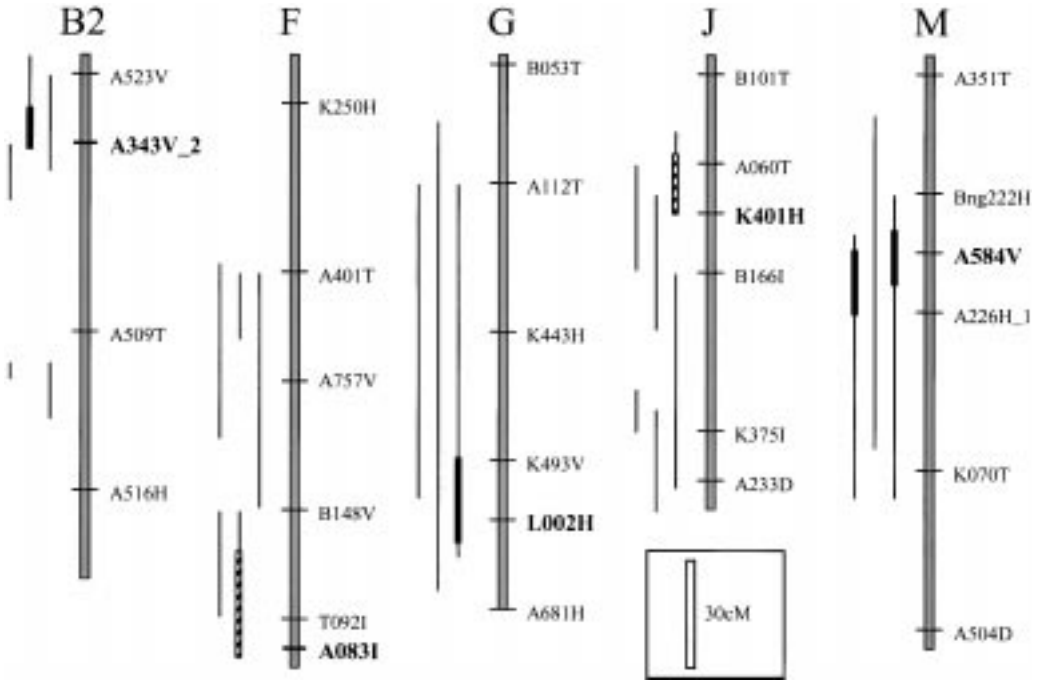

Fig. 2. Confidence intervals for corn earworm weight gain resistance QTLs in three soybean crosses. The large, shaded bars represent soybean linkage groups with LG designations adapted from the integrated soybean restriction fragment length polymorphism (RFLP)/simple sequence repeat (SSR) map (Cregan et al., 1999). RFLP markers in normal typeface are included from that map as evenly spaced reference points. RFLP markers listed in boldface are those that were found closest to the quantitative trait loci logarithm of the odds peak(s) and are specific to the maps constructed for this study. Vertical lines to the left of each linkage group approximate the portion(s) of each linkage group.

Only one antibiosis QTL was detected in Cross 1. A comparison of the R^2 value of this QTL (28%) and the heritability estimate in this cross (54%) suggests that other antibiosis QTLs exist in this cross. As discussed above, it is possible that PI171451 possesses the resistance allele that was detected on LG J in Cross 3. However, no QTL can be confidently detected in that region unless markers with normal segregation ratios can be mapped there. It may be necessary to do so in a separate cross of these two parents. While QTL detection was hampered in this population due to isolated regions of abnormal segregation, there is no indication that the abnormal segregation was associated in any way with insect resistance, especially since the large QTL on LG M was also detected in Cross 3.

The R^2 value from a multiple regression model including the two significant markers in Cross 2 (41%) is close to the heritability estimate ($h^2 = 42\%$) and suggests that the major QTLs for antibiosis have been detected in this cross, although R^2 estimates may be inflated in studies with small populations (Beavis, 1998). In Cross 3, the phenotypic variance accounted for by the three significant markers in a multiple regression model was $R^2 = 49\%$, which is lower than the heritability estimate ($h^2 = 62\%$). This suggests that there may be undetected QTLs remaining in this cross.

Recently, genetic maps of soybean have been made using new marker technologies such as amplified fragment length polymorphism (AFLP; Keim et al., 1997) and SSRs (Cregan et al., 1999). These new maps, anchored to RFLP frameworks, demonstrate the utility of these new markers in quickly developing maps with many markers covering large genomic regions. The sizes of the AFLP map (650 AFLP markers; >3400 cM) and the SSR map (600 SSR markers; >2800 cM) indicate that the maps used in this study could be improved by the use of these technologies. This would enable phenotypic variance for insect resistance to be more fully explained in these soybean genotypes.

Lambert and Kilen (1984b) suggested that PRI could be maximized by the incorporation of resistance genes from all three of these PIs into a single cultivar. Until now, this has not been considered feasible because of uncertainty about gene number estimates and the amount of linkage drag that would be encountered by incorporating PI171451, PI227687, and PI229358 into a single breeding program. In addition, the practice of assaying for only one form of PRI (i.e. antibiosis or antixenosis, but not both) in soybean breeding for this trait appears to have neglected significant amounts of heritable resistance. Through QTL analysis with RFLP maps we have identified markers linked to antibiosis QTLs in crosses of Cobb to each of these three PIs. These QTLs include two in which insect resistance was conditioned by the inheritance of the allele from Cobb, the susceptible parent. Comparison of soybean antibiosis QTLs with previously reported antixenosis QTLs (Rector et al., 1998, 1999) has demonstrated the partial genetic independence these two PRI components, in that of the nine QTLs detected among the four genotypes tested, only one QTL (on LG M) was detected for both antibiosis and antixenosis. These results suggest that the two modes of PRI should be considered as distinct traits in a soybean breeding program. Marker-assisted selection for PRI in soybean involving the most robust marker-QTL combinations reported here and previously should facilitate the pyramiding of insect resistance genes from Cobb, PI171451, PI227687, and PI229358.

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